ETIOLOGY OF ACUTE KIDNEY INJURY IN HOSPITALIZED PATIENTS IN A TERTIARY CARE HOSPITAL

Md. Ataul Karim^{1*} Md Habibur Rahman² Md Habib Hassan² A S M Zahed³ Md Jashim Uddin³ Shoman Sarkar³

Abstract

Background: Acute Kidney Injury (AKI) previously referred to as acute renal failure complicates acute care hospital admissions and intensive care unit patients. In AKI associated with sepsis and multiple organ failure,mortality is high and the outcome is usually determined bythe severity of the underlying disorder and other complications, rather than by kidney injury itself. Aim of our study was to evaluate the etiological profile of AKI in a tertiary care hospital like Chittagong Medical College Hospital (CMCH).

Materials and methods: This observational study was conducted among 100 hospitalized patients in different wards of CMCH from November 2018 to April 2019. Patients were selected based on the Kidney Disease Improving Global Outcomes (KDIGO) criteria of AKI. An increase in serum creatinine >0.3 mg/dl or >1.5 times the baseline and a fall of urine output <0.5 ml/kg/h for 6–12 h were the criteria for selection. A structured questionnaire was completed andbaseline level of serum creatinine, urine output were documented.

Results : Out of 100 patients, 56 were males and 44 were females. Mean age of the patients was 40.89±12.74 years. Diabetes mellitus and Hypertension were the common comorbidities. Hypovolaemia and Sepsis were found to be most common cause which included diarrheal illness (27 patients) followed by Urinary Tract Infection (9 patients) and Community-Acquired Pneumonia (4 patients). Eleven patients had Surgical causes and only 8 had Obstetrical causes.

- Assistant Registrar of Medicine Chittagong Medical College Hospital, Chattogram.
- 2. Assistant Professor of Medicine Chittagong Medical College, Chattogram.
- Associate Professor of Medicine Chittagong Medical College, Chattogram.

*Correspondence: Dr. Md. Ataul Karim

E-mail: md.a.karimcmc@gmail.com Cell: 01618 83 72 10

Submitted on : 13.07.2020 Accepted on : 26.07.2020 **Conclusion:** Causes of AKI are preventable and treatable. Timely appropriate management will reduce the incidence and improve the outcome of AKI.

Key words

Acute kidney injury; Kidney Disease Improving Global Outcomes; Renal function.

Introduction

Acute Kidney Injury (AKI) describes the situation where there is a sudden and often reversible loss of renal function, which develops over days or weeks and is often accompanied by a reduction in urine volume¹. AKI is defined as an absolute increase in serum creatinine by 0.3 mg/dL or more within 48 hours or a relative increase of greater than or equal to 1.5 times baseline that isknown or presumed to have occurred within 7 days. AKI is characterized as oliguric ifurine production is less than 400–500 mL/day or less than 20 mL/h². AKI may be community-acquired or hospital-acquired. Common causes of community-acquired AKI include volume depletion, heart failure, adverse effects of medications, obstruction of the urinary tract, or malignancy³. The most common clinical settings for hospital-acquired AKI are sepsis, major surgical procedures, critical illness involving heart or liver failure, and nephrotoxic medication administration³. Incidence of AKI has increased over the past few decades and it is variable indifferent regions of the world⁴⁻⁶. Reliable statistics on the patterns and prevalence of AKI in Bangladesh are not available, but AKI is the most common renal emergency encountered in Asian hospitals⁷. The study was designed to observe the etiology of AKI in admitted patients of Chittagong Medical College Hospital.

Materials and methods

This observational study was carried out on 100 admitted patients in different departments of Chittagong Medical College Hospital from 01/11/2018 to 30/04/2019.

Inclusion criteria

i. Patients of 18 years or more admitted in different wards of CMCH with AKI as evidenced by clinical features and biochemical markers

Exclusion criteria

- i. Chronic Kidney Disease (CKD)
- ii. Acute on Chronic Kidney Disease
- iii. Subjects who did not provide written consent to participate in the study.

After approval of protocol from Ethical Review Board, voluntary informed consent was taken from patients or attendants. A structured questionnaire was used to collect demographic information including age and sex. Patients were selected based on the Kidney Disease Improving Global Outcomes (KDIGO) criteria of AKI. Clinical data included primary diagnosis, past medical history, presence of co-morbidities, surgical status, physical examination findings, lab investigations, Baseline level of serum creatinine, urine output were documented. Eight ml of blood was drawn for Complete Blood Count, Blood Urea, Serum Creatinine and Urine is collected for routine and microscopic examination. Chest X-Ray, Plain X-Ray KUB, Ultrasonography done in relevant cases. Data were compiled in a Microsoft Office Excel Worksheet. SPSS (Statistical Package for Social Science) version 23 software is used to process and analyze the data. Continuous variables were reported as the means \pm SD and categorical variables were reported as frequency (Percentages). Data were presented in appropriate tables and charts.

Results

Out of 100 patients of AKI: 56% males and 44% females. Male to Female ratio 1.3: 1. Mean age of patients was 40.89±12.74. Most patients (74%) were from >31 years of age group. Only 12% of cases were under 20 years of age group (Table I).

Table I: Age and Sex distribution of the 100 hospitalized AKI patients.

Age in years	Male	Female	Total
<20 years	07 (12.5%)	05 (11.4%)	12 (12.0%)
21-30 years	08 (14.3%)	08 (18.2%)	16 (16.0%)
31-40 years	19 (33.9%)	18 (40.9%)	37 (37.0%)
≥41 years	22 (39.3%)	13 (29.5%)	35 (35.0%)
Total	56 (56.0%)	44 (44.0%)	100 (100%)
Mean ±SD	43.24±12.15	34.33±13.20	40.89±12.74

Majority of the patient were from urban or semiurban area (81%). Monthly family income <20,000 BDT in 59% patients. One fourth patients had educational qualification primary or below.Most of the male patients were involved in service or business. Majority female were housewives.

Figure 1 - Among the co-morbidity: Diabetes Mellitus (46%) Hypertension (25%) Ischaemic Heart Disease (22%) Chronic Obstructive Pulmonary Disease (20%). Pre-existing Chronic Liver Disease (5%) and Cough Variant Asthma (5%).

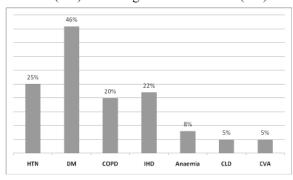


Fig 1: Prevalence of different co-morbidities in 100 hospitalized AKI patients.

Table II: Mode of Presentation of the 100 hospitalized AKI patients.

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Mod	le of presentation	Frequency	Percentage
1	Oliguria	76	(76%)
2	Loose motions	27	(27%)
3	Hypotension	13	(13%)
4	Drowsiness	13	(13%)
5	Fever	42	(12%)
6	Oedema	11	(11%)
7	Vomiting	09	(9%)
8	Anuria	09	(9%)
9	Shock	09	(9%)
10	Haematuria	08	(8%)
11	Burning micturition	07	(7%)
12	Hypertension	07	(7%)
13	Shortness of breath	06	(6%)
14	Loin Pain	04	(4%)
15	Convulsion	03	(3%)
16	Diabetic foot	02	(2%)

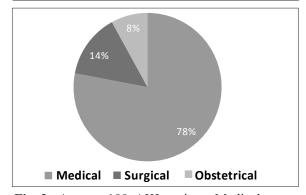


Fig 2: Among 100 AKI patients-Medical cases (78%) Surgical (14%) Obstetrical (8%).

Table III: Etiology of AKI in 100 hospitalized patients.

patients.		
Causes of AKI	Frequency*	Percentage
Sepsis		
Urinary tract infection	09	(9%)
Respiratory infections, Pneumonia	04	(4%)
Infected diabetic foot	02	(2%)
Hepatitis	02	(2%)
Rhabdomyolysis	01	(1%)
Hypovolemia		
Diarrheal illness	27	(27%)
Acute pancreatitis	04	(4%)
Chronic liver disease	05	(5%)
Burns	03	(3%)
Cardiac causes	04	(4%)
Organophosphorus compound poisoning	03	(3%)
Nephrotoxic drugs/chemicals	06	(6%)
Lupus nephritis	02	(2%)
Acute glomerulonephritis	08	(8%)
Surgical causes		
Obstructive uropathy	06	(6%)
Postoperative	05	(5%)
Obstetrical causes		
Puerperal sepsis	03	(3%)
Severe preeclampsia, Eclampsia	03	(3%)
Postpartum hemorrhage	01	(1%)
HELLP syndrome	01	(1%)

^{*}Findings overlap.

Table IV: Findings of Routine Urine Analysis of the 100 hospitalized AKI patients.

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Findings	Number of patients	Percentage
Normal	48	48%
Proteinuria	23	23%
WBC in urine	09	09%
RBC in urine	11	11%
RBC casts	08	08%
Granular casts	01	01%

^{*}Findings overlap

Routine urine analysis was Normal (48%) Protienuria (23%) WBC (9%) RBC (11%) and RBC cast (8%).

Table V: Renal Biochemistry of the 100 hospitalized AKI patients.

Parameter (unit)	Mean	Standard deviation
Blood Urea (mg/dl)	91	34
Serum Creatinine(mg/dl)	4.29	2.55
Serum Potassium (mmol/l)	4.44	1.69
Serum calcium (mg/dl)	8.6	1.5
Serum phosphate(mg/dl)	5.5	1.2
Serum parathyroid hormone(pmol/L)	3.9	1.1

Mean blood urea was 91±34 mg/dl whereas serum creatinine was 4.29±2.55 mg/dl at presentation.

Table VI: Other investigations profile of the 100 hospitalized AKI patients.

Investigations	*Findings	Frequency (%)
Complete Blood Count	Increased WBC	63 (63%)
Chest X-ray	Pulmonary oedema	11 (11%)
USG of KUB	Renal/ureteric calculi	05 (5%)
Plain X-Ray KUB	Renal/ureteric calculi	05 (5%)
Intravenous urogram	Obstruction	06 (6%)

^{*}Findings overlap.

Discussion

AKI is a heavy global burden associated with both short and long term mortality. It has been considered as a preventable and treatable disease⁸. In the present study, mean patients age was 40.89±12.74 years. Most patients (74%) were >31 years age group. This was similar to the findings of other study on Bangladeshi population but different from western population^{9,10}. As most patients has comorbidities, this age group is susceptible. Male to female ratio was 1.3: 1 in the present study which is consistent with Eswarappa et al while in Mostafi et al this was 3:2. Diabetes was the common comorbidity, followed by HTN, IHD and COPD^{11,9}. Vikrant et al reported chronic obstructive pulmonary disease, Hypertension, Coronary artery disease, Chronic liver disease, Diabetes Mellitus were the comorbid conditions¹². Soren et al observed Diabetes as the most common comorbidity followed by Hypertension which is consistent with our findings¹³. Oliguria was the common (76%) mode of presentation whereas 15% were nonoliguric and 9% were anuric. Mostafi et al reported 66% presented with oliguria and 10.47% with anuria⁹. We found diarrhoea 27%, fever 12%, edema 11% in our study. Delay in sufficient fluid supplement in the form of oral rehydration saline or intravenous fluid precipitates AKI. In this study hypovolemia mostly due to diarrhoea and vomiting was the leading cause (27%) of AKI. Mostafi et al and another Indian study reported 60% of AKI cases due to gastroenteritis^{9,14}. Sepsis due to urinary tract infection, respiratory infection are found next cause of AKI. Delayed diagnosis, insufficient fluid therapy, inappropriate antibiotics complicate AKI. Contrast-induced nephropathy is the third most common cause of AKI in western countries¹⁵. In the present study 6% cases were

due to nephrotoxic drugs or contrast induced. We found 5 cases of postoperative AKI, who had normal renal function preoperatively which is close to other study findings of 1.1-17% 16. We had 6 cases of AKI due to obstructive uropathy. Among pregnancy related AKI, we found puerperal sepsis, pregnancy induced hypertension, HELLP syndrome and postpartum hemorrhage which is dissimilar to otherreports¹⁷. Routine urine analysis was normal in 48% of the enrolled AKI patients. Protienuria, WBC and RBC in urine indicates nephropathy and urinary tract infection, which is consistent withMostafi et al9. In this study we have large number of pre-renal AKI cases which can be treated conservatively. Detail history, clinical examination and routine investigation will help to explore aetiology of AKI. Early reporting, immediate appropriate management will revert most AKI cases and improve their from worse outcome.

Limitations

- Small sample size and from a single center does not truly reflect the etiological pattern of AKI throughout the country
- ii. Sample was collected conveniently
- iii. Pediatric patients were not included.

Conclusion

Acute Kidney Injury (AKI) is an important clinical problem associated with worse outcome for hospitalized patients. Hypovolaemia and sepsis are found common prerenal causes of AKI. Biological toxins, Nephrotoxic drugs/chemicals, Lupus nephritis, Acute glomerulonephritis are the renal causes of AKI. Obstructive uropathy, Puerperal sepsis, Severe preeclampsia, Eclampsia, Postpartum hemorrhage are reported for etiology of post renal AKI.

Recommendations

Abundant data confirm that the incidence of AKI, particularly in hospitalized patients, is rising. Timely appropriate management will reduce the incidence and worse outcome of AKI. Large multicenter prospective study is needed to get the real etiological scenario of AKI throughout the country.

Acknowledgements

The authors gratefully acknowledge the contribution of all the doctors and staff working in the different department of Chittagong Medical College Hospital.

Contribution of authors

MAK-Conception, acquisition of data, drafting and final approval.

MHR- Acquisition of data, drafting and final approval.

MHH- Design analysis, interpretation of data and final approval.

ASMZ- Analysis, drafting and final approval.

MJU-Acquisition of data, drafting and final approval. SS -Design analysis, interpretation of data and final approval.

Disclosure

All the authors declared no competing interests.

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